

Constrictive pericarditis complicating an endocardial pacemaker

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SUMMARY A patient is described with constrictive pericarditis after insertion of an endocardial pacemaker. As far as we know this complication has not been reported previously.

Permanent endocardial pacemakers are now commonly implanted and a number of complications have been described.¹ Constrictive pericarditis has been reported as a complication of epicardial pacemakers²; this has never been reported, however, after insertion of an endocardial pacemaker.

Case report

A 62-year-old man was admitted with a three week history of episodic dizziness and effort dyspnoea. He had had mild hypertension for one year, but there was no history of any other cardiovascular disease.

On physical examination he had a heart rate of 35/minute, blood pressure of 235/90 mmHg, and a normal jugular venous pressure. The apex beat was mildly thrusting. On auscultation the heart sounds were normal and there was a grade 1/4 ejection systolic murmur in the aortic area.

The electrocardiogram showed 2:1 atrioventricular block and left anterior hemiblock with a ventricular

rate of 35 a minute. Chest x-ray film showed moderate cardiomegaly.

A transvenous demand pacemaker was inserted via the left subclavian vein. The lead, a Lucas wedgeless tip model CLI-L-002, was easily positioned at the apex of the right ventricle (Fig. 1a), with no complications. He remained well until four weeks later when he developed dyspnoea, dry cough, and ankle oedema. Examination now showed a pyrexia of 38.5°C, and sinus tachycardia with a small volume pulse showing severe paradox. The jugular venous pressure was raised 8 cm with a large "a" wave. The heart sounds were now muffled and in addition there was a third heart sound with a pericardial friction rub at the left sternal border. The pacemaker was found to be working normally.

The electrocardiogram showed sinus tachycardia with left bundle-branch block; the chest x-ray film showed a pronounced increase in heart size and no change in position of the pacemaker lead (Fig. 1b). An echocardiogram confirmed the presence of a large

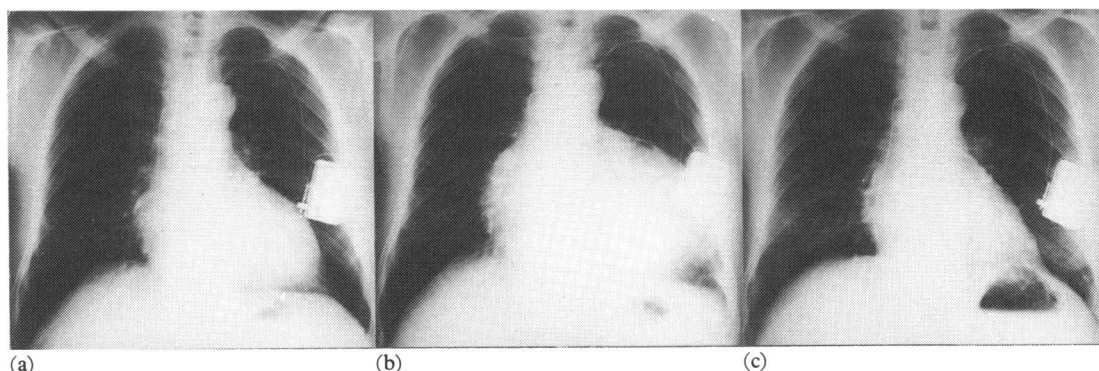


Fig. 1 Series of chest x-rays. (a) Two days after pacemaker insertion showing moderate cardiomegaly (unchanged from before implantation) and good electrode position. (b) Two months later showing large pericardial effusion. (c) One year later showing decrease in heart size shortly before pericardectomy.

pericardial effusion (Fig. 2a). Other investigations showed the following: Hb 10 g/dl, WBC 11.2, ESR 145 mm/hour (all previously normal). Antinuclear factor, cardiac antibodies, and viral studies were all negative.

Pericardial aspiration was performed with air replacement and limited right heart catheter studies (Table); 600 ml slightly bloodstained fluid was removed. Subsequent x-rays showed normal outline of the heart and pericardium. Examination of the pericardial fluid was sterile and negative for malignant

cells. There were no acid-fast bacilli on staining and culture.

On treatment with paracetamol and diuretics the patient's symptoms settled but the tachycardia and raised jugular venous pressure with tall "a" and steep "y" descent remained.

Ten months later he deteriorated, developing severe dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, cough, and ankle oedema. The only new feature on examination was that the heart sounds were more muffled. The jugular venous pressure remained raised with a steep "y" descent.

The chest x-ray film showed an obvious reduction in heart size (Fig. 1c) and was now smaller than before pacemaker insertion. The echocardiogram showed no evidence of a pericardial effusion (Fig. 2b). Cardiac catheterisation (Table) showed a raised right atrial pressure with a steep "y" descent and equal right and left ventricular diastolic pressure with a prominent "dip and plateau" configuration (Fig. 3).

The left ventricular angiogram was normal and the right ventricular angiogram showed an abrupt termination of diastolic filling. Selective coronary arteriograms were normal.

At thoracotomy the pericardium was found to be thickened and densely adherent, especially over the inferior surface of the right ventricle, and was extensively excised. Histology showed fibrous tissue with focal lymphocytic infiltration. Culture was negative for *Mycobacterium tuberculosis*. He has since made a good recovery, with relief of his symptoms.

Discussion

Shortly after insertion of this patient's pacemaker he developed acute pericarditis with effusion and sub-acute tamponade.³ He later developed pericarditis requiring pericardectomy.

Although it is possible that transient perforation of the right ventricular wall occurred at the time of

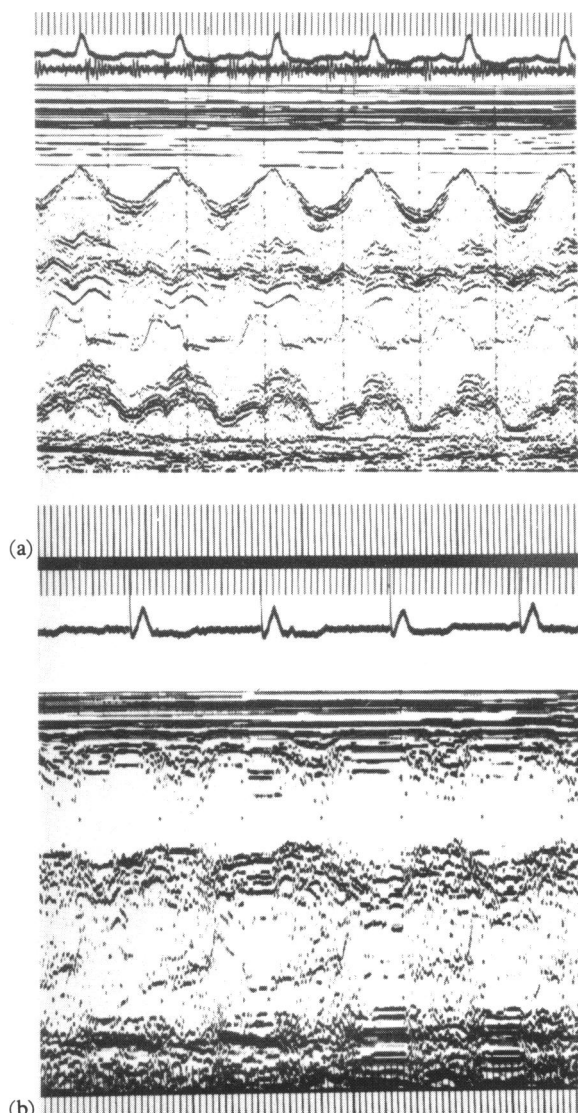


Fig. 2 Echocardiograms. (a) Showing large pericardial effusion. (b) Complete resolution of effusion.

Table Pressures taken before and after pericardiocentesis and at cardiac catheterisation

	Pressures (mmHg)		
	Before pericardial aspiration	After pericardial aspiration	At catheterisation
RA mean	10	5	10
LA/wedge* mean	17*		13
RV	32/15		24/12
LV			120/13
Ao/RFA	140/86	165/95	122/78
Mean	103	125	100

*Pulmonary wedge pressure; at second catheterisation the pressure was direct left atrial.

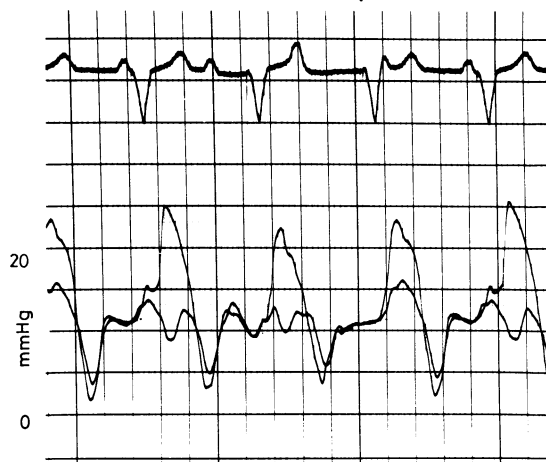


Fig. 3 Simultaneous right atrial and right ventricular pressure traces at cardiac catheterisation showing typical "dip and plateau" configuration of constrictive pericarditis.

implantation, there was never any evidence to suggest this, though it is thought to be a not infrequent occurrence. Perforation has been reported as causing acute tamponade³ but there has been no report of severe pericarditis or pericardial constriction after suspected perforation.

Even though the cardiac antibodies in this case were negative it was felt likely that the acute illness was Dressler's syndrome. This was supported by the time of onset after pacemaker insertion, the physical findings, and the low Hb, raised white cell count, and raised erythrocyte sedimentation rate. The investigations did not indicate any alternative cause for the pericarditis and subsequent constriction. There has been one previous report of a case of Dressler's syn-

drome occurring after insertion of an endocardial pacemaker,⁴ but this was not followed by any long-term complications. It is possible that if right ventricular perforation did occur the associated minor trauma to the myocardium could initiate Dressler's syndrome.

Constrictive pericarditis is unusual after Dressler's syndrome or the post-cardiotomy syndrome but it has been reported⁵ and has been seen in this Unit (unpublished observation).

References

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